Maternal smoking modulates fatty acid profile of breast milk

Agnieszka Szlagatys-Sidorkiewicz (aga1@gumed.edu.pl)[1], Dorota Martysiak-Żurowska[2], Grzegorz Krzykowski[3], Maciej Zagierski[1], Barbara Kamińska[1]

1.Department of Pediatrics, Pediatric Gastroenterology, Hepatology and Nutrition, Medical University of Gdansk, Gdansk, Poland
2.Department of Food Chemistry, Technology and Biotechnology, Chemical Faculty, Gdansk University of Technology, Gdansk, Poland
3.Institute of Mathematics, University of Gdansk, Gdansk, Poland

Keywords
Diet, Fatty acids, Human milk, Lactation, Smoking

DOI:10.1111/apa.12276

ABSTRACT

Aim: We hypothesized that the fatty acid composition of breast milk can be affected by a smoking habit in the mother. Consequently, this study verified whether maternal smoking modulates, and if so to what extent, the breast milk fatty acid profile.

Methods: The study included 20 postpartum women who declared smoking more than five cigarettes daily throughout a period of pregnancy and lactation, and 136 nonsmoking postpartum women. Breast milk samples were collected between the 17th and the 30th day after delivery. The samples were analysed by means of high-resolution gas chromatography for overall content of saturated, monounsaturated and polyunsaturated fatty acids.

Results: Compared with nonsmokers, smokers were characterized by significantly higher relative breast milk contents of fatty acids and monounsaturated fatty acids. Additionally, smokers' breast milk had higher concentrations of selected saturated fatty acids, monounsaturated fatty acids and polyunsaturated fatty acids. Furthermore, smokers were characterized by significantly lower values of linoleic to arachidonic acid ratio and n-6 to n-3 polyunsaturated fatty acids ratio.

Conclusions: Aside from its other harmful consequences, smoking modulates the fatty acid profile of human milk.

INTRODUCTION

Breast milk fats play a variety of biological functions. Aside from being a principal source of energy for the infant, they also constitute a vital component of cellular membranes and neural cells, as well as are the carriers of fat-soluble vitamins A, D, E and K. Moreover, they may have immunomodulatory functions (1).

Triglycerides, corresponding to approximately 98% of breast milk fats, are synthesized by the follicular cells of the mammary gland; the substrates include glycerol as well as fatty acids that are synthesized *de novo* or from diet- and adipose tissue-originating precursors. The remaining 2% of breast milk fats is comprised of phospholipids, cholesterol, di- and monoacylglycerols and free fatty acids (1).

Both the quantitative and qualitative content of breast milk fat are modulated by an array of factors. The quantity of breast milk fat is lactation phase specific and increases with the time of lactation (2). Furthermore, the breast milk content of fat is also influenced by the gestational age; the lower the age at birth, the higher the fat content (3). Although the overall quantity of breast milk fat is not modulated by the maternal diet, the latter markedly influences its fatty acid profile (4). Aside from the maternal diet, gestational age has also been shown to modify the fatty acid composition of human milk significantly: the milk from mothers of preterm infants differed from that of full-term newborns in terms of its medium- and long-chain polyunsaturated n-3 and n-6 fatty acid content (3). Additionally, the breast milk profile of fatty acids can also be modulated by the health status of the mother. Among others, changes in the fatty acid content were reported in mothers with insulin-dependent diabetes (5) and such chronic inflammatory processes as allergy (6) or cystic fibrosis (7).

Among an array of harmful effects, smoking can also induce chronic inflammation. An increased concentration of C-reactive protein has been reported in smokers along with elevated white blood cell count and higher levels of fibrinogen, IL-6 and prostaglandin F2-alpha (8). Furthermore, smoking-related changes of blood lipid profile have

Key notes

- This study shows that aside from its other harmful consequences, smoking modulates the fatty acid profile of human milk.
- Compared with nonsmokers, smokers are characterized by significantly higher relative breast milk contents of fatty acids and monounsaturated fatty acids.
- Furthermore, smokers show lower values of linoleic to arachidonic acid ratio and n-6 to n-3 polyunsaturated fatty acids ratio.

been documented, such as elevated triglyceride levels and a decreased concentration of HDL-cholesterol (9). Finally, alterations in fatty acid profile of the smokers' blood have been revealed (10).

Smoking during lactation modulates the composition of the human breast milk considerably as confirmed by the decreased contents of protein and total fat (9,11), the lower concentrations of docosahexaenoic acid (DHA, 22:6 [n-3]) (9) and sIgA (11), the altered cytokine profile (12), the reduced antioxidant potential (13) and the higher levels of lipid oxidation products generated during the reactive oxygen species-mediated reactions (14).

In view of this aforementioned evidence, we hypothesized that also the fatty acid composition of breast milk can be affected by a smoking habit in the mother. Consequently, this study verified whether maternal smoking modulates, and if so to what extent, the breast milk fatty acid profile.

METHODS

Participants

This case–control study was performed between January 2009 and August 2010 and included women who delivered at the Obstetrical Ward of the Pomeranian Center of Traumatology in Gdansk (Northern Poland) and met the following inclusion criteria: age above 18 years, no history of chronic disorders or pregnancy complications, normal full-term vaginal delivery (38th–40th gestational week), good general status of the neonate (8–10 Apgar points), normal birth weight (2500–4000 g) and exclusive breast-feeding. The exclusion criteria included passive smoking, acute and chronic disorders (such as gestational diabetes and atopy), pharmacotherapy or the use of supplements other than vitamins, including the preparations with n-3 fatty acid content.

Medical documentation of each pregnant woman referring to delivery was analysed, and if she satisfied the 'medical' enrolment criteria, her smoking status was determined with a structured interview. According to sparse and mostly anecdotal data, the fraction of Polish women who smoke during pregnancy can be as high as 20%. Considering the yearly number of deliveries per our unit (2000) and the period covered by this study (20 months), one would expect even 600 smokers in our material. However, in reality, this number was markedly lower. First of all, about one-third of pregnant women were excluded based on 'medical' criteria. Such high exclusion rate resulted from the tertiary character of obstetrical unit analysed and high fraction of complicated pregnancies. Consequently, 2235 women who met the 'medical' criteria were asked about their smoking status. Among them, there were 20 who declared smoking during pregnancy. Assuming 10–20% smoking rate among pregnant women in Poland, there could be 200–450 smokers in our sample and 20 cases correspond to 4.5–10% of this. Therefore, we qualified all smokers who met the eligibility criteria, which corresponded to every 10th to 20th women who theoretically smoked during pregnancy Considering the size of the control group, we have chosen seven nonsmokers per one identified smoker because we assumed that some of those who were not identified as smokers at a time of delivery could smoke earlier in pregnancy or resume smoking during lactation.

On the day of sampling, participating mothers completed a survey pertaining to their health status and overall condition of their infants, and active smoking during pregnancy and lactation. On the basis of that declaration, the participants were qualified into the smokers' group (Group I, at least five cigarettes daily throughout a period of pregnancy and lactation, n = 20) and the nonsmokers' group (Group II, n = 136). According to previous studies, such verbal declaration constitutes a sufficient and reliable tool to identify women who smoke during pregnancy and lactation (15,16).

Ethics

All the procedures were approved by the Local Ethics Committee of the Medical University in Gdansk. The subjects gave their written informed consent before the start of any procedure.

Breast milk sampling

Breast milk samples were collected between the 17th and the 30th day after delivery. The mammary gland was evacuated completely with the aid of an electric lactator 2 h after the first morning feeding. The milk was collected into sterile glass containers. After careful mixing, 5-mL samples were taken, placed into other sterile containers and immediately frozen at −80°C. The remaining milk was fed to the infants.

Dietary assessment

Participants' diet was analysed directly before the breast milk sampling, based on the 72-h nutritional diary reported by mothers. The data were analysed using Dieta 4.0 (National Institute of Food and Nutrition, Poland) package with regard to the nutritional value of average dietary daily intake and its contents of various nutrients, including its overall content of fat, as well as its content of saturated (SFA), monounsaturated (MUFA) and polyunsaturated (PUFA) fatty acids.

Analytical procedure

Breast milk fat was extracted using the Roese-Gottlieb method (17), which was modified for the purpose of this analysis: the stage of fat drying at 102°C was replaced by evaporation of the solvent at 40°C under reduced pressure. Nitrogen was then used to remove the residual solvent. The fatty acids of the extracted fat were then converted into fatty acid methyl esters (FAME) in accordance with the European Norm (18). Subsequently, FAMEs were divided according to the length of their hydrocarbon chain and the degree of their saturation by means of high-resolution gas chromatography (19), using the Hewlett-Packard gas chromatograph with split/splitless injector (1:100 ratio), flame ionization detector and Rtx 2330 column (100 m × 0.25 mm; Restek, Bellefonte, PA, USA).

Table 1 Demographic and obstetrical characteristics of study participants

Parameter	Group I (smokers, n = 20)	Group II (nonsmokers, n = 136)	p value
Age (years)	27.29 ± 5.66	30.04 ± 4.09	0.020
Parity (n)	2 (1–2.5)	2 (1–2)	0.450
Maternal weight at delivery (kg)	79.219 (62.87–90)	75.47 (69–82)	0.415
Gestational age (weeks)	40 (39–41)	40 (40–41)	0.130
Birth weight (g)	3558 (3228–3458)	3580 (3280–3521)	0.348
Apgar score at 1st min (points)	9.8 (9.5–10)	9.62 (9.5–10)	0.390
Feeds per day (n)	6 (6–8)	6 (6–7)	0.746
Declaration of smoking	20/20	0/136	–

Values presented as arithmetic means (±SD) or medians and interquartile ranges (in parenthesis).

Quantitative and qualitative analysis of the fatty acids was conducted against standard solutions of FAMEs (Supelco Bellefonte, PA, USA; Larodan Fine Chemicals, Malmö, Sweden) using external standard method with correction factors (19). The results were expressed as weight percentages of all identified fatty acids with 6- to 24-carbon chain lengths, containing up to six double bonds.

Statistical analysis

The normal distribution of continuous variables was tested with the Kolmogorow–Smirnov test. The results were presented as arithmetic means and their standard deviations, and intergroup differences in fatty acid contents were analysed with Student's test. Calculations were performed using Statistica 8 (StatSoft®, Tulsa, OK, USA) software and R Statistical Environment (20) with statistical significance defined as $p \leq 0.05$.

RESULTS

The characteristics of the study participants are presented in Table 1. The average age of the smoking women was significantly lower than that of the nonsmokers.

Dietary assessment

Smoking women were characterized by a lower average daily consumption of carbohydrates as compared to nonsmokers ($p = 0.032$); furthermore, they obtained significantly less energy from carbohydrates ($p = 0.038$) and more energy from fat ($p = 0.037$). Studied groups did not differ significantly in terms of fatty acid consumption (Table 2).

Table 2 Average dietary daily intakes of study participants

Parameter	Group I (smokers, n = 20)	Group II (nonsmokers, n = 136)	p value
Protein (g)	82.93 ± 31.44	79.92 ± 25.76	0.339
Fat (g)	76.55 ± 31.40	70.36 ± 26.91	0.199
Carbohydrates (g)	247.80 ± 73.61	281.89 ± 90.45	0.032
Energy from proteins (%)	16.81 ± 2.78	16.16 ± 4.11	0.180
Energy from fat (%)	34.07 ± 8.74	30.34 ± 7.46	0.037
Energy from carbohydrates (%	49.02 ± 8.91	53.50 ± 7.77	0.038
SFAs (g)	29.20 ± 13.65	26.91 ± 12.00	0.237
MUFAs (g)	30.12 ± 14.47	26.93 ± 11.56	0.172
PUFAs (g)	11.59 ± 6.02	10.09 ± 5.73	0.146
Sum of fatty acids (g)	70.90 ± 28.90	63.92 ± 25.36	0.152
SFAs (% of fatty acids)	41.42 ± 9.75	42.02 ± 7.77	0.394
MUFAs (% of fatty acids)	41.36 ± 6.86	41.96 ± 5.35	0.352
PUFAs (% of fatty acids)	17.22 ± 8.64	16.02 ± 6.47	0.273

Values presented as arithmetic means (±SD).

Table 3 Total fat, fatty acid, SFA, MUFA and PUFA breast milk contents (weight percentages) in study participants

Parameter	Group I (smokers, n = 20)	Group II (nonsmokers, n = 136)	p value
Total fat (per 100 g of milk)	2.78 ± 1.56	3.00 ± 1.54	0.272
Total SFAs (% of total fatty acids)	42.01 ± 3.01	42.63 ± 4.55	0.212
Total MUFAs (% of total fatty acids)	43.88 ± 2.57	42.72 ± 3.35	0.039
Total PUFAs	13.44 ± 1.48	13.75 ± 2.20	0.211
PUFAs to SFAs ratio	0.32 ± 0.053	0.33 ± 0.08	0.306

SFA, saturated fatty acid; MUFA, monounsaturated fatty acid; PUFA, polyunsaturated fatty acid. Values presented as arithmetic means (±SD).

Human milk fatty acid composition

Total contents of fat, fatty acids, SFAs, MUFAs and PUFAs are presented in Table 3. A higher content of fatty acids expressed as a percentage of total fat was found in the milk of smoking mothers as compared to that of nonsmokers ($p = 0.031$). Similarly, smokers were characterized by a significantly higher content of MUFAs ($p = 0.039$).

Saturated fatty acids (SFAs)

Both in smoking and nonsmoking mothers, SFAs represented about 42% of all breast milk fatty acids. Palmitic acid (16:0) was the most prevalent SFA in both groups (approximately 24.5%), followed by stearic acid (18:0, about 6% each group). Smokers' breast milk was characterized by

Table 4 Saturated fatty acid breast milk contents (weight percentages per 100 g of fatty acids) in study participants

Saturated fatty acid (common name)	Group I (smokers, n = 20)	Group II (nonsmokers, n = 136)	p value
Caproic acid 6:0	0.03 ± 0.01	0.03 ± 0.02	0.281
Caprylic acid 8:0	0.13 ± 0.05	0.14 ± 0.07	0.153
Capric acid 10:0	1.04 ± 0.34	1.15 ± 0.45	0.105
Undecylic acid 11:0	0.04 ± 0.05	0.04 ± 0.03	0.275
Lauric acid 12:0	3.87 ± 1.25	4.42 ± 1.45	0.040
Tridecylic acid 13:0	0.03 ± 0.01	0.08 ± 0.44	0.132
Myristic acid 14:0	4.82 ± 1.03	5.50 ± 1.47	0.013
Pentadecylic acid 15:0	0.33 ± 0.15	0.34 ± 0.20	0.347
Palmitic acid 16:0	24.52 ± 2.42	24.23 ± 2.23	0.308
Margaric acid 17:0	0.30 ± 0.05	0.30 ± 0.07	0.449
Stearic acid 18:0	6.61 ± 1.25	6.13 ± 1.03	0.060
Arachidic acid 20:0	0.18 ± 0.08	0.19 ± 0.04	0.299
Behenic acid 22:0	0.07 ± 0.06	0.06 ± 0.02	0.283
Lignoceric acid 24:0	0.07 ± 0.08	0.07 ± 0.08	0.349

Values presented as arithmetic means (±SD).

Table 5 Monounsaturated fatty acid breast milk contents (weight percentages per 100 g of fatty acids (% by weight of total fattyacids ±SD) in study participants

Monounsaturated fatty acid	Group I (smokers, n = 20)	Group II (nonsmokers, n = 136)	p value
Triadecenoic acid 13:1	0.11 ± 0.04	0.10 ± 0.10	0.442
Myristoleic acid 14:1 (n-5)	0.21 ± 0.08	0.22 ± 0.09	0.256
Other 14:1 isomers (*cis* + *trans*)	0.16 ± 0.14	0.15 ± 0.19	0.320
Sum of 15:1 (*cis* + *trans*)	0.08 ± 0.03	0.10 ± 0.04	0.038
Palmitoleic acid 16:1 (n-7)	2.50 ± 0.98	2.48 ± 0.69	0.484
Other 16:1 isomers	0.28 ± 0.11	0.31 ± 0.15	0.136
Sum 17:1 (*cis* + *trans*)	0.23 ± 0.14	0.22 ± 0.08	0.412
Oleic acid 18:1 (n-9)	35.36 ± 2.19	34.17 ± 3.18	0.039
Vaccenic acid 18:1 11t	2.41 ± 0.49	2.28 ± 0.37	0.146
Gadoleic acid 20:1(n-9)	0.65 ± 0.21	0.60 ± 0.17	0.164
Erucic acid 22:1 (n-9)	0.14 ± 0.09	0.13 ± 0.07	0.199
Nervonic acid 24:1(n-9)	0.13 ± 0.17	0.08 ± 0.06	0.140

Values presented as arithmetic means (±SD).

significantly higher concentrations of lauric (12:0, p = 0.04) and myristic acid (14:0, p = 0.01; Table 4).

Monounsaturated fatty acids (MUFAs)

In both studied groups, MUFAs corresponded to approximately 42–43% of all breast milk fatty acids. Oleic acid (18:1 [n-9]) was the most prevalent MUFA of human milk; the concentration of that acid was significantly higher in smokers than that in nonsmokers (p = 0.03). Moreover, smokers were characterized by a significantly higher content of group 15:1 MUFAs (the sum of *cis* and *trans* isomers, p = 0.03; Table 5).

Polyunsaturated fatty acids (PUFAs)

Linoleic acid (LA, 18:2 [n-6]) corresponded to approximately 10%, while alpha-linolenic acid (ALA, 18:3 [n-3]) constituted about 1% of all breast milk fatty acids in both examined groups. Smokers were characterized by significantly higher concentrations of eicosadienoic acid (EDA, 20:2 [n-6], p = 0.029) and arachidonic acid (AA, 20:4 [n-6], p = 0.043; Table 6).

Table 7 summarizes overall breast milk contents of n-3 and n-6 fatty acids. Significantly higher concentrations of all n-6 fatty acids except LA were documented in smokers as compared to nonsmokers (p = 0.03). Furthermore, smokers were characterized by significantly lower values of LA to ALA ratio and n-6 to n-3 ratio.

DISCUSSION

Maternal smoking exerts multivariate harmful effects both during the foetal stage and the extra-uterine life. Neonates of mothers who smoked during pregnancy are not only characterized by a lower birth weight, but their body composition is also altered as suggested by a reduced lean body mass (21). In contrast, an important question whether maternal smoking during lactation can modulate the body composition of the infant during the postnatal period has not been verified thus far. Mothers who smoke decide on breastfeeding less frequently, and the duration of their lactation is significantly shorter (22). Furthermore, postnatal maternal smoking represents the principal risk factor of respiratory infections in the neonate (23). Potential routes of exposure to tobacco smoke components during neonatal period and infancy include passive smoking, contact with tobacco smoke residues (e.g. on parental and infant clothing, bedding, household items, etc.) and direct transfer via the mother's breast milk. However, the question whether, and to what extent, the breast milk composition is directly altered by maternal smoking rather than to be modified secondarily to different lifestyle characteristics of female smokers is still opened.

In this study, we have observed significant differences in the dietary characteristics of the analysed groups: smokers were characterized by a lower average consumption of carbohydrates and a higher ingestion of dietary fats. However, those differences did not correspond to different consumption levels of long-, medium- and short-chain fatty acids. In contrast, previous studies did not document smoking-related differences in dietary contents of fats and carbohydrates (24) and instead revealed that smokers are characterized with a higher ingestion of SFAs and a lower consumption of PUFAs (25). Additionally, the smokers' diet is characterized by a reduced consumption of fibre, vitamins and minerals (24). Plausibly, all those differences can be attributed mostly to the inhomogeneity of the studied populations in terms of their lifestyles, age and general health.

In our study, the average age of smoking women was significantly lower than that of nonsmokers. Also, other authors have similar observations with regard to the age of women who smoke during pregnancy and lactation (21). It is unlikely, however, that such a slight difference of age (2.75 years on average) could considerably confound the other findings of this study.

Table 6 Polyunsaturated fatty acid breast milk contents (weight percentages per 100 g of fatty acids,% by weight of total FAs ±SD) in study participants

Polyunsaturated fatty acids	Group I (smokers, n = 20)	Group II (nonsmokers, n = 136)	p value
Conjugated linoleic acid, isomers 18:2 (*cis trans*)	0.31 ± 0.08	0.32 ± 0.10	0.355
n-6 fatty acids			
Linoleic acid 18:2 (n-6)	9.37 ± 1.57	10.00 ± 1.91	0.056
Gamma-linolenic acid 18:3 (n-6)	0.10 ± 0.06	0.06 ± 0.04	0.130
Eicosadienoic acid 20:2 (n-6)	0.38 ± 0.19	0.29 ± 0.10	0.029
Dihomo-gamma-linolenic acid 20:3 (n-6)	0.40 ± 0.14	0.36 ± 0.10	0.109
Arachidonic acid 20:4 (n-6)	0.54 ± 0.17	0.47 ± 0.15	0.043
Docosadienoic acid 22:2 (n-6)	0.10 ± 0.08	0.08 ± 0.05	0.156
Adrenic acid 22:4 (n-6)	0.04 ± 0.03	0.04 ± 0.03	0.493
Docosapentaenoic acid 22:5 (n-6)	0.25 ± 0.23	0.22 ± 0.21	0.313
n-3 fatty acids			
Alpha-linolenic acid 18:3 (n-3)	1.23 ± 0.37	1.17 ± 0.47	0.274
Eicosatrienoic acid 20:3 (n-3)	0.08 ± 0.05	0.07 ± 0.06	0.303
Eicosapentaenoic acid 20:5 (n-3)	0.06 ± 0.03	0.07 ± 0.10	0.140
Docosapentaenoic acid 22:5 (n-3)	0.18 ± 0.05	0.19 ± 0.12	0.119
Docosahexaenoic acid 22:6 (n-3)	0.29 ± 0.12	0.33 ± 0.10	0.074

Values presented as arithmetic means (±SD).

Table 7 Contents of n-3 and n-6 polyunsaturated fatty acid breast milk contents (weight percentages per 100 g of fatty acids) in study participants

Parameter	Group I (smokers, n = 20)	Group II (nonsmokers, n = 136)	p value
Linoleic acid (LA)	9.37 ± 1.57	10.00 ± 1.91	0.056
n-6 PUFAs except LA	1.90 ± 0.67	1.59 ± 0.42	0.030
Sum of all n-6 PUFAs	11.27 ± 1.33	11.59 ± 1.95	0.173
Alpha-linolenic acid (ALA)	1.23 ± 0.37	1.17 ± 0.47	0.274
n-3 PUFAs except ALA	0.64 ± 0.18	0.62 ± 0.26	0.339
Sum of all n-3 PUFAs	1.87 ± 0.42	1.80 ± 0.57	0.239

Values presented as arithmetic means (±SD).

This study documented an array of smoking-related differences in the breast milk profile of fatty acids. Smokers' milk was characterized by lower concentrations of certain SFAs: lauric and myristic acid, as well as the group of C15:1 MUFAs (expressed as the sum of *cis* and *trans* isomers). In contrast, the breast milk from smokers had a significantly higher concentration of oleic acid. Interestingly, other authors have reported a higher concentration of myristic acid, palmitoleic acid and oleic acid in smokers' plasma as compared to that of nonsmokers (10).

Most smoking-related changes pertained to the breast milk content of PUFAs. This group of fatty acids has an array of biological functions, among which, they build cellular membranes and constitute precursors for biologically active inflammatory modulators: eicosanoids and docosanoids (leukotrienes, prostaglandins, lipoxins and resolvins). We did not observe differences with regard to the breast milk concentrations of essential fatty acids (LA and ALA) in the studied groups. According to the literature, breast milk concentrations of those acids and their metabolites correspond to their dietary contents (26). We did not observe significant differences in the amount of ALA (n-3) family metabolites, both overall and in the case of individual acids. In contrast, studied groups differed significantly in terms of LA family (n-6) derivative contents. The breast milk of smoking women was characterized by a higher relative overall content of all n-6 PUFAs except LA, as well as by greater concentrations of eicosadienoic acid and arachidonic acid. To the best of our knowledge, only one study dealing with the effects of maternal smoking on the breast milk contents of PUFAs has been published thus far. It did not reveal any significant smoking-related differences in the relative contents of LA, ALA and AA (% by weight FA), both on the 1st day of lactation and 1, 3 and 6 months thereafter. However, the same study documented a decrease in the absolute levels of LA, ALA and AA after 1 month of lactation (9). This latter finding points to the necessity of further studies of longitudinal changes of fatty acid profile occurring throughout lactation. The same authors observed that smokers were characterized by significantly lower relative (after 3 months of lactation) and absolute (in lactation months 1 and 3) concentrations of DHA (9). This phenomenon was confirmed by our findings because we observed a trend of decreasing DHA content in breast milk in smoking women. This lower breast milk concentration of DHA in smokers is reflected by a lower supply of this acid to infants (9). This raises the question about the necessity to supplement babies from smoking mothers with DHA.

The blood concentration of PUFAs reflects the dietary content of those acids and the variance of their genetically controlled metabolism (27). Irrespective of the reason, the levels of eicosapentaenoic acid (EPA, 20:5 [n-3]), DHA and AA in the blood of smokers are lower than in those of nonsmokers (10).

An important issue undoubtedly requiring further investigation is the question of the potential biological consequences of the smoking-related shift towards a higher

content of n-6 fatty acids in the PUFA profile that was documented in our study. While maternal consequences of exposure to tobacco smoke are quite obvious, one can ask to what extent breast milk can mediate those harmful effects to the infant. Moreover, the role of breast milk PUFA profile in this potential mediatory effect is unclear. Nevertheless, previous studies revealed the association between the PUFA composition of human milk and the prevalence of allergies in children. Higher colostrum level of docosapentaenoic acid was associated with increased risk of atopic eczema, and higher total level of n-3 acids in mature milk was associated with higher risk of nonatopic eczema in the infant. Finally, higher colostrum levels of n-6 fatty acids corresponded to the increased risk of rhinitis (28). Furthermore, the PUFA profile was proved to modulate both the growth rate of preterm newborns (29) and the mental development of all infants (30).

CONCLUSION

To the best of our knowledge, this study is the first to analyse such a wide spectrum of breast milk fatty acids with regard to a maternal smoking status. We observed several significant smoking-related differences of the breast milk composition, pertaining to the level of PUFAs as well as to that of SFA and MUFA concentrations. Although further research is required to understand the biological significance of those differences in fatty acid composition, one can conclude that aside from its other harmful consequences, smoking modulates the fatty acid profile of human milk.

References

1. Agostoni C, Marangoni F, Grandi F, Lammardo AM, Giovannini M, Riva E, et al. Earlier smoking habits are associated with higher serum lipids and lower milk fat and polyunsaturated fatty acid content in the first 6 months of lactation. *Eur J Clin Nutr* 2003; 57: 1466–72.
2. Koletzko B, Agostoni C, Bergmann R, Ritzenthaler K, Shamir R. Physiological aspects of human milk lipids and implications for infant feeding: a workshop report. *Acta Paediatr* 2011; 100: 1405–15.
3. Bitman J, Wood L, Hamosh M, Hamosh P, Mehta NR. Comparison of the lipid composition of breast milk from mothers of term and preterm infants. *Am J Clin Nutr* 1983; 38: 300–12.
4. Molto-Puigmarti C, Castellote AI, Carbonell-Estrany X, Lopez-Sabater MC. Differences in fat content and fatty acid proportions among colostrum, transitional, and mature milk from women delivering very preterm, preterm, and term infants. *Clin Nutr* 2011; 30: 116–23.
5. Peng Y, Zhou T, Wang Q, Liu P, Zhang T, Zetterstrom R, et al. Fatty acid composition of diet, cord blood and breast milk in Chinese mothers with different dietary habits. *Prostaglandins Leukot Essent Fatty Acids* 2009; 81: 325–30.
6. Jackson MB, Lammi-Keefe CJ, Jensen RG, Couch SC, Ferris AM. Total lipid and fatty acid composition of milk from women with and without insulin-dependent diabetes mellitus. *Am J Clin Nutr* 1994; 60: 353–61.
7. Johansson S, Wold AE, Sandberg AS. Low breast milk levels of long-chain n-3 fatty acids in allergic women, despite frequent fish intake. *Clin Exp Allergy* 2011; 41: 505–15.
8. Bitman J, Hamosh M, Wood DL, Freed LM, Hamosh P. Lipid composition of milk from mothers with cystic fibrosis. *Pediatrics* 1987; 80: 927–32.
9. Wannamethee SG, Lowe GD, Shaper AG, Rumley A, Lennon L, Whincup PH. Associations between cigarette smoking, pipe/cigar smoking, and smoking cessation, and haemostatic and inflammatory markers for cardiovascular disease. *Eur Heart J* 2005; 26: 1765–73.
10. Simon JA, Fong J, Bernert JT Jr, Browner WS. Relation of smoking and alcohol consumption to serum fatty acids. *Am J Epidemiol* 1996; 144: 325–34.
11. Bachour P, Yafawi R, Jaber F, Choueiri E, Abdel-Razzak Z. Effects of smoking, mother's age, body mass index, and parity number on lipid, protein, and secretory immunoglobulin A concentrations of human milk. *Breastfeed Med* 2012; 7: 179–88.
12. Ermis B, Yildirim A, Tastekin A, Ors R. Influence of smoking on human milk tumor necrosis factor-alpha, interleukin-1beta, and soluble vascular cell adhesion molecule-1 levels at postpartum seventh day. *Pediatr Int* 2009; 51: 821–4.
13. Zagierski M, Szlagatys-Sidorkiewicz A, Jankowska A, Krzykowski G, Korzon M, Kaminska B. Maternal smoking decreases antioxidative status of human breast milk. *J Perinatol* 2012; 32: 593–7.
14. Ermis B, Yildirim A, Ors R, Tastekin A, Ozkan B, Akcay F. Influence of smoking on serum and milk malondialdehyde, superoxide dismutase, glutathione peroxidase, and antioxidant potential levels in mothers at the postpartum seventh day. *Biol Trace Elem Res* 2005; 105: 27–36.
15. McDonald SD, Perkins SL, Walker MC. Correlation between self-reported smoking status and serum cotinine during pregnancy. *Addict Behav* 2005; 30: 853–7.
16. Pickett KE, Rathouz PJ, Kasza K, Wakschlag LS, Wright R. Self-reported smoking, cotinine levels, and patterns of smoking in pregnancy. *Paediatr Perinat Epidemiol* 2005; 19: 368–76.
17. Association of Official Analytical Chemists. Method No 905.02. *Official methods of analysis*. 19th ed. Washington: Association of Official Analytical Chemists (AOAC), 1990.
18. International Standard ISO 5509:2000. Animal and vegetable fats and oils. Preparation of methyl esters of fatty acids, 2000.
19. International Standard ISO 5508:1990. Animal and vegetable fats and oils. Analysis by gas chromatography of methyl esters of fatty acids.
20. R Development Core Team. *R: a language and environment for statistical computing*. Vienna: R Foundation for Statistical Computing, 2011.
21. Samper MP, Jimenez-Muro A, Nerin I, Marqueta A, Ventura P, Rodriguez G. Maternal active smoking and newborn body composition. *Early Hum Dev* 2012; 88: 141–5.
22. Gerd AT, Bergman S, Dahlgren J, Roswall J, Alm B. Factors associated with discontinuation of breastfeeding before 1 month of age. *Acta Paediatr* 2012; 101: 55–60.
23. Jones LL, Hashim A, McKeever T, Cook DG, Britton J, Leonardi-Bee J. Parental and household smoking and the increased risk of bronchitis, bronchiolitis and other lower respiratory infections in infancy: systematic review and meta-analysis. *Respir Res* 2011; 12: 5.
24. Midgette AS, Baron JA, Rohan TE. Do cigarette smokers have diets that increase their risks of coronary heart disease and cancer? *Am J Epidemiol* 1993; 137: 521–9.
25. Thompson RL, Pyke S, Scott EA, Thompson SG, Wood DA. Cigarette smoking, polyunsaturated fats, and coronary heart disease. *Ann N Y Acad Sci* 1993; 686: 130–9.
26. Nasser R, Stephen AM, Goh YK, Clandinin MT. The effect of a controlled manipulation of maternal dietary fat intake on

medium and long chain fatty acids in human breast milk in Saskatoon, Canada. *Int Breastfeed J* 2010; 5: 3.
27. Lattka E, Rzehak P, Szabo E, Jakobik V, Weck M, Weyermann M, et al. Genetic variants in the FADS gene cluster are associated with arachidonic acid concentrations of human breast milk at 1.5 and 6 mo postpartum and influence the course of milk dodecanoic, tetracosenoic, and trans-9-octadecenoic acid concentrations over the duration of lactation. *Am J Clin Nutr* 2011; 93: 382–91.
28. Lowe AJ, Thien FC, Stoney RM, Bennett CM, Hosking CS, Hill DJ, et al. Associations between fatty acids in colostrum and breast milk and risk of allergic disease. *Clin Exp Allergy* 2008; 38: 1745–51.
29. Tinoco SM, Sichieri R, Setta CL, Moura AS, Carmo MG. n-3 polyunsaturated fatty acids in milk is associate to weight gain and growth in premature infants. *Lipids Health Dis* 2009; 8: 23.
30. Auestad N, Scott DT, Janowsky JS, Jacobsen C, Carroll RE, Montalto MB, et al. Visual, cognitive, and language assessments at 39 months: a follow-up study of children fed formulas containing long-chain polyunsaturated fatty acids to 1 year of age. *Pediatrics* 2003; 112: e177–83.